# Important Advances in Clinical Medicine

## Epitomes of Progress—Chest Diseases

*The Scientific Board of the California Medical Association presents the following inventory of items of progress in chest diseases. Each item, in the judgment of panel of knowledgeable physicians, has recently become reasonably firmly established, both as to scientific fact and important clinical significance. The items are presented in simple epitome and an authoritative reference, both to the item itself and to the subject as a whole, is generally given for those who may be unfamiliar with a particular item. The purpose is to assist the busy practitioner, student, research worker or scholar to stay abreast of these items of progress in chest diseases which have recently achieved a substantial degree of authoritative acceptance, whether in his own field of special interest or another.*

*The items of progress listed below were selected by the Advisory Panel to the Section on Chest Diseases of the California Medical Association and the summaries were prepared under its direction.*

Reprint requests to: Division of Scientific and Educational Activities, California Medical Association, 731 Market St., San Francisco, CA 94103

## Asbestosis

ASBESTOSIS is a pneumoconiosis resulting from the inhalation of asbestos particles, and manifested by diffuse interstitial fibrosis of the lung and fibrotic reactions of the parietal pleura. It is found predominantly in workers directly involved with asbestos, such as miners, millers of asbestos, shipyard workers and insulation workers. There are, however, more than 4,000 known uses of asbestos fiber and production has risen from 50 tons in 1877 to over 4 million tons in 1970. It is prudent for clinicians to become familiar with common work-classifications and industries associated with exposure to asbestos. There is also an estimated emission into the environmental air of 50,000 tons per year with emission in California (in 1966) of about 1,500 tons. Asbestosis has even been diagnosed in nonoccupational groups, particularly in household contacts of asbestos workers and also in groups living near asbestos production or use sources.

In addition to producing pulmonary and pleural fibrosis, it has been strongly associated pathologically with bronchogenic carcinoma, with mesotheliomas of the pleura and peritoneum, and probably with some gastrointestinal cancer. Physiologically, it has resulted in restrictive manifestations, decrease in gas transport measurements and some small airways dysfunction, with subsequent development of cor pulmonale.

It is of particular concern that clinical manifestations of asbestosis as well as malignant sequelae may not become evident for more than 20, 30 or 40 years after exposure. Malignancy in persons who worked in shipyards during World War II, many of whom reside along the West Coast, is just now becoming evident. Bronchogenic carcinoma is less usual in nonsmokers exposed to asbestos. In persons exposed to both asbestos and cigarette smoking the risk of cancer is significantly greater than additive. Those workers who have been exposed to asbestos fibers must be emphatically warned that smoking is an *extreme* hazard for them.

Symptoms of asbestosis do not differ from those of diffuse interstitial fibroses caused by other etiologic agents. Early breathlessness on exertion progresses to increasing dyspnea, even at rest. Cough is common with or without sputum and is often croupy in nature. Associated bronchitis from concomitant smoking is often found. Physical findings usually show somewhat characteristic crepitations on auscultation of the lower lung fields and less frequently in mid-lung fields. Digital clubbing is frequent. Radiographically,

the interstitial fibrosis is not diagnostic but characteristic pleural reactions are seen early. The presence of linear pleural or diaphragmatic calcifications should alert one to the possible past asbestos exposure.

"Restrictive" pulmonary function measurements (reduced vital capacity, reduced maximum voluntary ventilation) are occasionally accompanied by decreased forced expiratory volumes and impaired small airways measurements. Interference with gas transport is shown by reduced diffusing capacity of the lung for carbon monoxide (DLCO) as well as increased alveolar arterial gradient for oxygen pressure, which is worsened during exercise. Carbon dioxide retention is a late manifestation.

Asbestosis should be strongly suspected when an occupational detective search is initiated in the presence of dyspnea; radiologic evidence of fibrotic, pulmonary or pleural disease decreased spirometric measurements, decreased DLCO and hypoxemia. Asbestos bodies may be found occasionally in the sputum of asymptomatic persons. Presumptive diagnoses may be confirmed by the presence of asbestos fibers in biopsy specimens of lung tissue.

GERSCHEN L. SCHAEFER, MD

**REFERENCES**

Selikoff IJ, Bader RA, Bader ME, et al: Asbestosis and neoplasia. Am J Med 42:487-496, Apr 1967

Becklake MR: Asbestos-related diseases of the lung and other organs: Their epidemiology and implications for clinical practice. Am Rev Respir Dis 114(I):187-227, Jul 1976

## PEEP and Barotrauma

POSITIVE END-EXPIRATORY PRESSURE (PEEP) is now widely used as a therapeutic modality for acute respiratory failure (shock lung, diffuse capillary blood syndrome and respiratory distress syndrome). PEEP reduces shunt by preventing atelectasis, the primary cause of hypoxemia in this syndrome. This is accomplished by the associated increase in functional residual capacity (FRC). PEEP may also reduce shunt by discouraging the accumulation of alveolar and interstitial edema fluid. Improvement in the distribution of ventilation with PEEP has also been noted, which may be related to improved airway patency accompanying the increase in FRC as well as to alveolar recruitment with more uniform distribution of pulmonary compliance. The improvement in arterial oxygen tension with PEEP is often dramatic and permits rapid reduction in fraction of inspired oxygen ($FIO_2$) to safer levels.

PEEP may be accompanied by several adverse sequelae. The increase in mean transpulmonary pressure (more positive) may result in diminished venous return, and increased pulmonary vascular resistance resulting in reduced cardiac output and tissue oxygen supply. This is particularly likely with hypovolemic states although the administration of fluids in acute respiratory failure must be undertaken with great care. The use of intermittent mandatory ventilation (IMV) with PEEP or continuous negative chest pressure (cuirasse ventilator) instead of PEEP may be useful in avoiding these cardiovascular problems, although adequate respiratory support using these techniques is not always possible.

Barotrauma—including pneumothorax, subcutaneous and mediastinal emphysema—often accompanies the use of PEEP, although it is not clear whether this is more the consequence of the use of high inspiratory pressures and volumes than of PEEP itself. With preexisting hyperinflation, barotrauma is much more likely and PEEP is therefore contraindicated in obstructive lung diseases.

Appropriate and tolerable levels of PEEP are still being defined. PEEP levels of 5 to 15 cm of water are often adequate to effect a substantial improvement in hypoxemia without cardiovascular impairment or undue risk of barotrauma. These levels of PEEP have been associated with optimal static pulmonary compliance and maximum oxygen transport ("best PEEP"). In contrast, maximal improvement in shunt without detrimental effects on cardiac output ("optimal PEEP") has been noted with PEEP levels exceeding 40 cm of water. These undobutedly represent more severe varieties of acute respiratory failure in which cardiovascular effects and barotrauma are minimized because the higher pressures are not transmitted across the pulmonary parenchyma. Unfortunately in neither of the above studies was peak inspiratory pressure (and therefore degree of inspiratory inflation) controlled; therefore, it remains unclear whether PEEP alone was responsible for the improvement in oxygen supply. Nonetheless, it is probable that PEEP levels in excess of 15 cm of water may occasionally be necessary to produce optimal improvement in tissue oxygen delivery and perhaps in pulmonary compliance as well.

In practice PEEP should be limited to levels that are not associated with reductions in mixed venous oxygen tension or with serious cardiac output impedance, and which are sufficient to permit reduction of fraction of inspired oxygen to levels approximating 0.5 or less. These goals are now more readily attainable with pharmacologic car-